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Search - update  
L/cock 4/18/07

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(FILE 'HOME' ENTERED AT 11:17:14 ON 18 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 11:17:37 ON 18  
APR 2007

L1 364 S (MYOFILAMENT PROTEIN)  
L2 2 S L1 AND (MUSCLE DAMAGE)  
L3 2 DUPLICATE REMOVE L2 (0 DUPLICATES REMOVED)  
L4 6191 S (MUSCLE DAMAGE)  
L5 2 S L1 AND L4  
L6 29 S L4 AND ADDUCT?  
L7 4 S L6 AND TROPONIN?  
L8 3 S L7 NOT L5  
L9 1 DUPLICATE REMOVE L8 (2 DUPLICATES REMOVED)  
L10 13 DUPLICATE REMOVE L6 (16 DUPLICATES REMOVED)  
L11 12 S L10 NOT L5  
L12 11 S L11 NOT L9  
L13 3 S L12 AND PD<2000  
L14 8 S L12 NOT L13  
L15 4543 S (PROTEIN ADDUCT)  
L16 6 S L15 AND L4  
L17 2 DUPLICATE REMOVE L16 (4 DUPLICATES REMOVED)  
L18 202 S L4 AND TROPONIN?  
L19 93 S L18 AND PD<2000  
L20 47 DUPLICATE REMOVE L19 (46 DUPLICATES REMOVED)  
L21 0 S L20 AND ADDUCT?  
L22 2 S L20 AND TNI?  
L23 0 S L20 AND TNC  
L24 0 S L20 AND TNC?  
L25 0 S (TNI? ADDUCT)  
L26 0 S (TROPONIN ADDUCT?)

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L24 0 S L20 AND TNC?  
L25 0 S (TNI? ADDUCT)  
L26 0 S (TROPONIN ADDUCT?)

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AN 2000:809166 CAPLUS  
DN 134:111508  
ED Entered STN: 19 Nov 2000  
TI Experimental heart muscle damage in alcohol feeding is associated with increased amounts of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts  
AU Worrall, Simon; Richardson, Peter J.; Preedy, Victor R.  
CS Alcohol Research Unit, Department of Biochemistry, The University of Queensland, Brisbane, QLD 4072, Australia  
SO Addiction Biology (2000), 5(4), 421-427  
CODEN: ADBIFN; ISSN: 1355-6215  
PB Carfax Publishing  
DT Journal  
LA English  
CC 4-7 (Toxicology)  
AB Chronic and excessive alc. consumption induces defined myocardial lesions characterized by impaired structural, mech. and biochem. features. The pathogenic mechanisms are unknown, although it is possible that protein adduct formation by reactive metabolites of ethanol may be a contributory process. Hitherto, this has only been tested with respect to antibodies against reduced-acetaldehyde protein adducts in clin. studies, despite the fact that during alc. toxicity the formation of reduced-acetaldehyde, unreduced-acetaldehyde, malondialdehyde, malondialdehyde-acetaldehyde and hydroxyethyl protein adducts have been reported in non-cardiac tissues. It was the author's hypothesis that the heart is particularly sensitive to the formation of protein adducts in alc. toxicity. To test this hypothesis, the authors analyzed hearts from rats fed nutritionally complete liquid diets containing ethanol as 35% of total calories for 6 wk, using the Lieber-DeCarli pair-feeding protocol. Control rats were treated identically and fed the same diet in which ethanol was replaced by isocaloric glucose. At the end of the feeding period, the hearts were dissected and ventricular muscle analyzed. After 6 wk' ethanol feeding, ELISA anal. showed increased amts. of reduced-acetaldehyde protein adducts ( $p < 0.01$ ) unreduced-acetaldehyde ( $p < 0.01$ ) and malondialdehyde-acetaldehyde ( $p = 0.01$ ) protein adducts. However, malondialdehyde and  $\alpha$ -hydroxyethyl- protein adducts were not significantly increased in hearts of ethanol-fed rats compared to pair-fed control ( $p > 0.1$  in both instances). This is the first report of acetaldehyde adduct formation in alc. cardiomyopathy. This suggests that either immune process may develop or functional impairment of affected proteins may occur.  
ST heart muscle damage alc acetaldehyde malonaldehyde protein adduct; ethanol cardiotoxicity oxidative stress heart acetaldehyde malonaldehyde protein adduct; lipid peroxidn heart acetaldehyde malonaldehyde protein adduct  
IT Proteins, specific or class  
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)  
(adducts; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)  
IT Heart, disease  
(cardiomyopathy; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)  
IT Toxicity  
(cardiotoxicity; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with

- increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT Immunoassay  
(enzyme-linked immunosorbent assay; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT Heart  
Immunity  
Oxidative stress, biological  
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT Peroxidation  
(lipid; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT Heart  
(toxicity; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT 64-17-5, Ethanol, biological studies  
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)  
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT 75-07-0, Acetaldehyde, biological studies  
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)  
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)
- IT 75-07-0D, Acetaldehyde, adducts with proteins, biological studies  
542-78-9D, Malondialdehyde, adducts with proteins  
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)  
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD  
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AN 1996:62716 BIOSIS  
 DN PREV199698634851  
 TI Myosin heavy-chain fragments and cardiac troponins in the serum  
 in rhabdomyolysis: Diagnostic specificity of new biochemical markers.  
 AU Lofberg, Mervi [Reprint author]; Tahtela, Riitta; Harkonen, Matti; Somer,  
 Hannu  
 CS Dep. Neurol., Helsinki Univ. Central Hosp., 00290 Helsinki, Finland  
 SO Archives of Neurology, (1995) Vol. 52, No. 12, pp. 1210-1214.  
 CODEN: ARNEAS. ISSN: 0003-9942.  
 DT Article  
 LA English  
 ED Entered STN: 9 Feb 1996  
 Last Updated on STN: 10 Feb 1996  
 AB Background: Myosin is the major structural protein in muscle. Antibodies  
 to beta-type heavy meromyosin react with cardiac and slow-twitch skeletal  
 muscle. Cardiac TnT and TnI were developed as tissue-specific  
 indicators. Objectives: To study myosin heavy-chain fragments as a  
 delayed marker of previous rhabdomyolysis. To examine the cardiac  
 specificity of cardiac troponin T (TnT) and cardiac  
 troponin I (TnI) in patients with severe skeletal  
 muscle damage. Design and Methods: Serum myosin  
 heavy-chain fragments, TnT, and TnI were studied up to 12 days  
 after diagnosis in relationship to the serum creatine kinase level in 20  
 patients with rhabdomyolysis. The mean peak serum creatine kinase  
 activity was 91 300 U/L. Myosin heavy-chain fragments were measured by an  
 immunoradiometric assay, TnT by a one-step immunoenzymometric assay, and  
 TnI by an immunoenzymometric assay. Results: Values for serum  
 myosin heavy-chain fragments were greater than the upper limit of normal  
 in all patients. The peak value (70 times the upper normal limit, on  
 average) was usually achieved 4 to 7 days after the diagnosis of  
 rhabdomyolysis, and it was increased up to 12 days. The peak level of TnT  
 was increased in 95% of the patients, and it correlated strongly with the  
 peak activity of serum creatine kinase. The highest TnI value was above  
 the detection limit of myocardial infarction in 30% of the patients. Half  
 of these patients were the only patients with ischemic changes observed on  
 an electrocardiogram performed on admission to the hospital. Conclusions:  
 The measurement of myosin heavychain fragments was useful in the diagnosis  
 of previous rhabdomyolysis up to 12 days. The role of TnT was negligible  
 as an indicator of cardiac muscle damage in patients  
 with severe rhabdomyolysis. Cardiac TnI is a more  
 tissue-specific marker for myocardial damage even with concurrent  
 rhabdomyolysis.  
 CC Mathematical biology and statistical methods 04500  
 Clinical biochemistry - General methods and applications 10006  
 Biochemistry studies - Proteins, peptides and amino acids 10064  
 Biochemistry studies - Minerals 10069  
 Enzymes - Physiological studies 10808  
 Pathology - Diagnostic 12504  
 Metabolism - Minerals 13010  
 Metabolism - Proteins, peptides and amino acids 13012  
 Cardiovascular system - Physiology and biochemistry 14504  
 Cardiovascular system - Heart pathology 14506  
 Cardiovascular system - Blood vessel pathology 14508  
 Blood - Blood and lymph studies 15002  
 Blood - Blood, lymphatic and reticuloendothelial pathologies 15006  
 Muscle - Physiology and biochemistry 17504  
 Muscle - Pathology 17506  
 Nervous system - Physiology and biochemistry 20504  
 Nervous system - Pathology 20506  
 Public health - Public health administration and statistics 37010  
 Public health - Health services and medical care 37012  
 IT Major Concepts  
 Blood and Lymphatics (Transport and Circulation); Cardiovascular

Medicine (Human Medicine, Medical Sciences); Cardiovascular System (Transport and Circulation); Clinical Chemistry (Allied Medical Sciences); Enzymology (Biochemistry and Molecular Biophysics); Hematology (Human Medicine, Medical Sciences); Mathematical Biology (Computational Biology); Metabolism; Muscular System (Movement and Support); Nervous System (Neural Coordination); Neurology (Human Medicine, Medical Sciences); Pathology; Public Health (Allied Medical Sciences)

IT Chemicals & Biochemicals

CREATINE KINASE

IT Miscellaneous Descriptors

BETA-TYPE HEAVY MEROMYOSIN; CARDIAC MUSCLE DAMAGE;  
CARDIAC TROPONIN I; CARDIAC TROPONIN T; CREATINE  
KINASE; ISCHEMIA; MAJOR STRUCTURAL PROTEIN; MYOCARDIAL INFARCTION;  
NEUROLOGY; SEVERE SKELETAL MUSCLE DAMAGE;  
SLOW-TWITCH SKELETAL MUSCLE; STATISTICAL ANALYSIS

ORGN Classifier

Hominidae 86215

Super Taxa

Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

human

Taxa Notes

Animals, Chordates, Humans, Mammals, Primates, Vertebrates

RN 9001-15-4 (CREATINE KINASE)

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AN 1998152608 EMBASE  
TI Cardiac troponin-I is a marker of perioperative myocardial damage during cardiac surgery.  
AU Contini G.A.; Albertini D.; Antonelli A.M.; Campodonico R.; Corsanini E.; Grattagliano C.; Reverberi C.; Medici D.; Contini S.A.; Fragnito C.; Barbosa G.  
CS Dr. G.A. Contini, Divisione Cartedra Cardiochirurgia, Azienda Ospedaliera di Parma, Via Gramsci 14, 43100 Parma, Italy  
SO Journal of Cardiovascular Diagnosis and Procedures, (1998) Vol. 15, No. 1, pp. 41-45. .  
Refs: 15  
ISSN: 1073-7774 CODEN: JCDPE  
CY United States  
DT Journal; Article  
FS 018 Cardiovascular Diseases and Cardiovascular Surgery  
029 Clinical Biochemistry  
LA English  
SL English  
ED Entered STN: 2 Jun 1998  
Last Updated on STN: 2 Jun 1998  
AB Diagnosis of perioperative myocardial infarction is a challenging task. Clinical symptoms, electrocardiographic changes, and biochemical markers have inherent limitations as diagnostic tools; isoenzymes of creatine kinase, for instance, are of limited specificity because of skeletal muscle damage. Cardiac troponin I (cTnI) is considered by many investigators to be more cardiospecific than CK-MB. The aim of this study was to assess, with serial measurements of levels of cTnI and mass CK-MB, the incidence and importance of cardiac injury and its relation to the length of extracorporeal circulation and aortic cross-clamp time in patients undergoing cardiac surgery. Serial measurements of cTnI using the Stratus® TnI assay which employs monoclonal antibodies specific for the TnI cardiac isotype and mass CK-MB have been performed in seventy consecutive patients operated on for coronary bypass surgery or valve replacement. All the patients have been operated on by different surgeons, with standard extracorporeal and surgical procedures; myocardial protection was achieved by hematic cardioplegy, either cold intermittent or warm intermittent or continuous. After operation they were admitted in a dedicated intensive care unit with monitoring facilities. Baseline measurements of cTnI were below 0.6 ng/ml ( $0.129 \pm 0.422$ ), and rose slightly in normally convalescing patients, with peak values between 6 hours and the first postoperative day. Serum levels of cTnI rose in patients with myocardial injury and in those with longer extracorporeal circulation and aortic cross-clamp times.  
CT Medical Descriptors:  
\*heart muscle injury: CO, complication  
\*heart muscle injury: DI, diagnosis  
\*extracorporeal circulation  
\*aorta clamping  
\*heart surgery  
coronary artery bypass surgery  
heart valve replacement  
time  
heart protection  
cardioplegia  
heart disease: SU, surgery  
heart infarction: CO, complication  
heart infarction: DI, diagnosis  
human  
male  
female  
major clinical study